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Editorial

Chronic Stable Angina-Where do we go from here?

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Editorial

In the United States (US) a person dies every minute from coronary heart disease (CHD). In addition, every twenty-five seconds they have a coronary event. CHD accounts for 16% of all deaths, numbering 388,000 in 2010 [1]. It is the leading and second leading cause of death in the black and white populations, respectfully. Moreover, as our population ages in the US, the incidence of this disease continues to rise. It is clear that modern medical interventions have reduced the numbers of events over the past decade. However, as these types of interventions, medical and otherwise become more advanced the cost of managing CHD has increased accordingly.

Acute coronary syndromes (ACS) vary dramatically in their pathophysiology, evolution and clinical presentation from chronic coronary artery stenosis. This paradigm has driven huge advances in the approach to management of ACS including mechanical revascularization. With respect to chronic stenosis, our current practice guidelines direct us to approach this clinical entity in a much different fashion [2]. Firstly, risk assessment for our patients with angina pectoris is critical because it allows us to tailor medical therapy with the end goal of reduction in death and myocardial infarction (MI). If a resting electrocardiogram (ECG) can be interpreted and the patient can exercise adequately, an exercise stress test is preferred as the first step. The test can then be coupled with or without imaging depending on the pretest likelihood of disease. Results suggestive of high-risk coronary lesions should proceed towards an invasive approach. The definition is unclear with respect to this type of finding. There are some commonly accepted high-risk features that include transient left ventricular dilation, ST-segment elevation, abnormal DUKE treadmill score (< -11) and moderate or large areas of jeopardized viable myocardium (JVM) by nuclear imaging [3].

Initial risk stratification directs us to impact the disease process, reducing events coupled with symptom management. The cornerstone of disease modulation includes statin therapy an area of medical therapy that is currently undergoing a paradigm shift. The tools for event reduction include beta-blockade and angiotensin converting enzyme inhibitors. Symptom management for chronic stable angina (CSA) is achieved through vasodilation with calcium channel blockade and nitrates. Altering life style behavior plays an important role in the management of CHD in the form of smoking cessation, weight reduction and physical activity.

If symptoms cannot be controlled with aggressive medical therapy revascularization can be pursued. Currently revascularization in stable CHD is reserved for symptom relief but there is an ongoing discussion concerning survival benefit and/or event reduction. In the COURAGE trial medically optimized subjects were randomized to either percutaneous coronary intervention (PCI) or not with the investigators focusing their endpoints on reduction of cardiovascular (CV) events [4]. All subjects underwent coronary angiogram to clarify disease extent and then randomized. Of note only 30% of the randomized subjects had proximal left anterior descending artery (LAD) lesions. There was no difference in the composite endpoint of death from any cause and non-fatal MI; however there was an increase in revascularization in the medical arm. An additional study isolating the diabetic population to determine the effects of mechanical revascularization produced similar findings to COURAGE. In this high risk subject population, BARI 2D showed no difference in rates of death or CV events between PCI and medical therapy [5].

Many argue that anatomic assessment does not portend physiologic significance. Functional testing in the form of non-invasive stress testing has helped us risk profile our patients. However, it is still not entirely clear as to the clinical significance of much of these abnormal tests. Invasive functional testing seems to correlate well with an abnormal noninvasive test with positive and negative predictive values of 100 and 88%, respectively [6]. The problem with this correlation was that an abnormal test was not defined and as such it was difficult to truly identify the extent of ischemic tissue. In the FAME study revascularization was driven by angiography versus functionally significant or fractional flow reserve (FFR) abnormal lesions in multi-vessel CHD including subjects with ACS [7]. Moreover, this was not a study of medical management of CSA and maximized medical therapy. By intervening on this diverse patient population events were reduced including death, MI and repeat revascularization. There was no difference in functional status at one year. This study showed promise in addressing functionality as a means of impacting clinical outcomes. Following the FAME study we were left with the question of how to apply FFR to our population with stable CHD.

The FAME 2 study attempted to address this question. Subjects with CSA in who PCI was considered were enrolled in this trial [8]. The composite primary endpoint was reduced and it appeared that most of this difference was driven by urgent revascularization as there was no difference in death or MI. Of the subjects who underwent revascularization only half had objective evidence of ischemia upon presentation. Two-thirds of all subjects had received proximal or mid LAD stents suggesting larger areas of myocardium at risk and this may have accounted for the reduction in events.

In certain patient populations with specific angiographic and potentially functional coronary lesions there does not appear to be a difference in hard clinical endpoints. After many years and a number of eloquently produced studies we are still left with questions about

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how to manage our patients with CSA. The guidelines suggest that management of high-risk lesions should include consideration of coronary revascularization with the intent of improving survival. Is a high risk lesion one that produces at least a moderate amount of JVM and does increasing amounts of JVM correlate with increasing risk of coronary events? The larger aim of the ongoing ISCHEMIA trial is to answer this question. Already there are questions about how FFR-positive lesions will impact clinical decision making within this trial and how these decisions may affect outcomes.

In the future, we may be utilizing a number of different risk stratification tools to care for our patients. Development of complex algorithms involving both non-invasive and invasive testing, utilizing information from functional, anatomic and intravascular imaging modalities may ultimately assist in making difficult clinical decisions.

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